Paraspinal Muscle Response to Electrical Vestibular Stimulation

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Galvanic (electrical) vestibular stimulation (GVS) has been used to study the role of the vestibular system in postural control by inducing postural sway in standing subjects. The purpose of this study was to determine the timing and pattern of activation in the paraspinal muscles in response to GVS and to compare these responses with those in the muscles of the lower leg. Binaural-bipolar GVS was applied to the skin overlying the mastoid processes of 10 subjects while they stood on a force plate with their eyes closed. The stimulus consisted of a 0.6 mA 5-pulse sequence. Each pulse lasted for 2 s, followed by 4 s of rest. The centre of pressure (COP) vs. time for each trial was calculated from the reaction forces and moments. Surface electromyographic (EMG) signals from the paraspinal and gastrocnemius muscles were recorded bilaterally. The EMG signals were rectified and integrated (iEMG). The iEMG from the muscles on the cathodal side of the body were then subtracted from the iEMG of the anodal side muscles, to yield a differential EMG (dEMG). Both the paraspinal and gastrocnemius muscles became activated in response to the stimulus. The pattern of activation was consistent with the changes observed in the centre of pressure. The primary response in both muscles acted to move the body toward the anode. This primary response began at 74 ± 20 ms in the paraspinal muscles and at 118 ± 18 ms in the gastrocnemius. A second component of the response began at 232 ± 27 ms in the paraspinal muscles and 262 ± 54 ms in the gastrocnemius muscles. This second phase of the response was opposite in direction to the primary response and was responsible for decelerating the body and maintaining the deviated position of the centre of mass over the base of support. Following the termination of the stimulus, the opposite pattern of muscle activation in both the paraspinal and the gastrocnemius muscles was observed. The results of this study suggest that the paraspinal muscles may play a significant role in the frontal plane response to vestibular stimulation during stance in humans. Key words: electromyography, galvanic stimulus, paraspinal muscles, posture, vestibular.

INTRODUCTION

A number of tests are available for the assessment of patients with dizziness of suspected vestibular origin. The various tests each play a specific role in assisting diagnosis and guiding treatment of these disorders. Standard bedside tests to evaluate vestibular function, such as the Romberg test and the Fukuda stepping test, are useful in the detection of vestibulospinal disorders, but do not provide localizing or quantitative information. Dynamic posturography provides quantitative information, but involves sensory systems other than vestibular system. Furthermore, it relies on the measurement of the position of the centre of pressure (COP), which is assumed to reflect the position of the centre of mass (COM) of the body, but in fact is a function of numerous additional factors (1). In contrast, galvanic vestibular stimulation (GVS) elicits a vestibulospinal response by stimulating only the vestibular afferents. It has traditionally used the centre of pressure as its output measure (2, 3) and thus has been only moderately successful. However, it is also possible to measure either the position of the segments of the body in space (from which the location of the COM can be calculated) or the surface electromyographic (EMG) signal from muscles that are assumed to be activated by vestibulospinal efferents. In our laboratory, the measurement of the position of the head in space in response to GVS has been used clinically (4). Unfortunately, these measurements require specialized equipment that is not readily available, whereas, the equipment to measure surface of EMGs is readily available in many clinical settings. The challenge is to find a muscle that is directly activated in the vestibulospinal response and the activation of which is easily measured by surface EMG.

A number of investigators have examined the postural sway response to bipolar-binaural galvanic vestibular stimulation (the stimulation of both mastoids with currents of opposite polarities) with the head facing forward (5–7). Day (5) described the response as a “leaning and bending of the body towards the anodal ear”. Leaning of the body presumably occurs by changing the ankle angle, whereas bending could occur either by rotation of the pelvis (through the action of the adductors and abductors of the legs) or by curvature of the lower back (through the action of the paraspinal muscles). They demonstrated that the relative tilt between the torso and the pelvis accounts for approximately two-thirds of the angular deviation of the body. Many muscles are involved in controlling the position of the torso relative to the pelvis and could potentially effect a change in the torso angle. The paraspinal or erector spinae muscles (iliocostalis, longissimus and spinalis muscles) play an obvious role in controlling the sagit-
tal plane position of the torso, but also have the potential to play an important role in the control of posture in the frontal plane. This potential role in the frontal plane response to GVS makes the paraspinal muscles an excellent candidate as a muscle from which the vestibulospinal response can be measured. Further support for the idea of a vestibulospinal galvanic test employing the paraspinal EMG response comes from a study by Storper and Honrubia (8). They examined the EMG response to a bipolar-binaural sinusoidal galvanic stimulus in the Triceps Surae and concluded that the TS EMG is not a direct response of the vestibulospinal system, but was the result of the response to the vestibulospinal activation in more proximal muscles. Therefore, the measurement of truncal or cervical EMGs might lead to a more direct measure of vestibulospinal output. The goal of this study is to elucidate the role of the paraspinal muscles in the frontal plane response to galvanic vestibular stimulation and to compare it with the well-documented response in the muscles of the lower leg.

MATERIALS AND METHODS

Ten subjects (7 male, 3 female), aged 20–35 years, provided informed written consent to participate in this study. None of the subjects had a history of vestibular, neurological or musculoskeletal impairment, nor did any report a history of trauma or ototoxic drug intake. Vestibuloculartesting was not performed on the subjects. All of the subjects were naïve to the hypothesis of the study and were instructed to stand comfortably with their eyes closed, hands across the chest, and bare feet together.

Stimulus

Stimulating electrode sites were located on skin of the forehead and overlying the mastoid processes. An anaesthetic cream was applied to the sites for 5 min and then removed. Conductive rubber electrodes 20 mm in diameter (InVivo Metric, E352, Heraldsburg, CA, USA) were then applied to the skin at all three sites. A conductive-adhesive gel was used between skin and electrode surface (InVivo Metric E447 Tac gel). A linear stimulus isolator (World Precision Instruments, A395, Sarasota, FL, USA) under computer control was used to create the stimulus. A 0.6 mA bipolar-binaural stimulus (anode at one mastoid, cathode at the other) was employed. The stimulus consisted of a 5-pulse sequence preceded and followed by 5 s without stimulus. Each pulse lasted for approximately 2 s, followed by 5 s of rest. Four trials of each polarity and 4 null trials (no stimulus) were conducted. The order of presentation of the 12 trials was randomized.

Recording

EMG recording electrodes (Motion Control Inc., Salt Lake City, UT) were placed on the skin overlying the right and left paraspinal muscles at approximately the level of the first lumbar vertebra, 4 cm from the midline. EMG electrodes were also placed on the skin overlying the medial heads of both the left and right gastrocnemius muscles. The EMG signal was pre-amplified 350 times at the recording site, then amplified (the level of amplification was chosen so as to utilize the entire range of the D/A converter), sampled at 500 Hz and stored on a digital computer. The reaction forces and moments at the foot-floor interface were recorded through the use of a force-plate (Bertec 4060A paired with Bertec Mod AM6.2 Amplifier). The signals from the force-plate were fed to a bridge amplifier and then sampled at 500 Hz and stored for later analysis. The creation of the stimulus and the recording of the response were coordinated through the use of LabVIEW software (National Instruments, Inc.) and a National Instruments multifunction IO plug in card.

Analysis

In the analysis of the data, the responses were grouped according to the direction of the induced sway. Since subjects sway toward the anode (positive stimulus), this is termed the ipsilateral side of the body, whereas the cathodal side (negative stimulus) is termed the contralateral side of the body. This nomenclature is consistent with that previously employed in reporting findings of the postural sway response to monaural galvanic stimulation (4).

The centre of pressure vs time for each trial was calculated from the reaction forces and moments. The COP from the left positive stimulus (left trials) and the right positive (right trials) were averaged for each subject. The COP vs time from each of the subjects was then averaged together. The left and the right trials were then combined by plotting the COP vs time in the ipsilateral and contralateral directions.

Baseline levels of EMG (mean and standard deviation) for each of the muscles for each subject were calculated from the null trials. Normalized EMG signals were calculated by subtracting the mean and dividing by the standard deviation. This was done to remove the effect of the signal’s offset and gain, thus allowing the signals to be compared across subjects. The normalized EMG signals were squared, integrated with an exponential decay (50 ms time constant), and then square rooted. This quantity was termed the integrated EMG (iEMG). The contralateral iEMG was then subtracted from the ipsilateral iEMG, to demonstrate the potential combined effect of a “push-pull” two-muscle mechanism. This quan-
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In order to quantify the timing of the muscle activation in response to the galvanic stimulus, the values of a number of time parameters were determined. These values were determined from the difference between the dEMGs for anode left and anode right trials. This method of parameterization is similar to that used by Britton and Day (9) where they extracted the parameters from superimposed tracings of the responses to anode right and anode left stimulation. Four time points were determined. The first parameter (T1) corresponds to the time at which the two signals diverge. The second parameter (T2) is determined at the time that the first peak difference between the signals is observed. The third parameter (T3) is determined when the signals cross for the second time. Finally, the fourth parameter (T4) occurs at the second peak difference between the signals. The value of each of the parameters was calculated from the mean of the responses for each subject. Thus in the statistical analysis, each subject constitutes one observation and the standard error of the mean (SEM) is a reflection of the inter-subject variability.

RESULTS

Representative raw data for a single trial are shown in Figure 1. The response to the stimulus is evident in the lateral COP and in the paraspinal EMGs. The deviation in the lateral COP is larger than that in the antero-posterior COP and appears to be synchronized with the stimulus. The right and left paraspinal muscles become activated in opposition to each other and in synchrony with the stimulus.

The integrated and then averaged EMG for a representative subject is shown in Figures 2 and 3. During anode left stimulation there is an initial burst of activity in the left paraspinal muscle and an inhibition of the activity in the right paraspinal muscle (between 2 and 2.5 s in Figs 2a and b). This is followed by a reduction in the activity of the left paraspinal muscle and an increase in the activity of the right paraspinal muscle (between 2.5 and 3.5 s in Figs 2a and b). During anode right stimulation, the opposite responses occur in both muscles (Figs 2c and 2d). This pattern of response was consistent with observed changes in the lateral COP (as demonstrated in Fig. 1). The initial paraspinal muscle activation produces a lateral deviation of the torso towards the anode. The reversal of this pattern, the secondary activation, is required to bring the torso to rest in its deviated position and to maintain the tilted posture. The gastrocnemius iEMG activity (Fig. 3) is not as clearly seen as the paraspinal activity. Note that the right and left paraspinal muscles are activated in opposite patterns suggesting that the differential activation of the muscles (as quantified by the dEMG) is the relevant quantity and it will thus be used throughout the remainder of the results.

The latency measures T1-T5 were obtained from the difference of the left and right stimulus trials as described in the methods. Figure 4 shows the paraspinal dEMG data from one subject averaged across stimuli and grouped by stimulus polarity. This relative activation (dEMG) accentuates the responses shown in Figure 2. The time points (T1–T5) were determined as described in the methods and are shown for this subject in Figure 4. The average latency measurements for all subjects combined are shown in Table I. The standard error in Table I reflects the inter-subject variability in these measurements. T1, T2 and T3 were measured from the initiation of the stimulus, whereas T4 and T5 were

![Figure 1](image_url)
measured from the termination of the stimulus. The offset latencies (T4 and T5) were larger than the onset latencies, but these differences were not significant due to the large variability in the offset measures. Note that the onset of the response (T1) in the paraspinal muscles preceded the onset of the response in the gastrocnemius muscles. The relative timing in the activation of the two muscle groups can be more clearly seen in Figure 5, which shows the difference between the latencies for the parameters T1–T3 for each of the subjects.

The ensemble averaged (across all subjects, and stimuli) dEMGs and lateral COP are shown in Figure 6. These averaged traces show paraspinal responses similar to those shown in Figure 4 for the example subject. In addition, the averaged gastrocnemius shows a clear pattern of activation that was not visible in the single subject traces. The pattern of activation in the gastrocnemius is similar to the pattern observed in the paraspinal muscles, but it is opposite in direction. Thus, the initial response is activation of the contralateral gastrocnemius and inhibition on the ipsilateral side and the secondary response is ipsilateral activation and contralateral inhibition. The pattern of displacement of the centre of pressure vs time is consistent with the pattern of activation of the paraspinal and gastrocnemius muscles. It can be seen that changes in activation in the paraspinal muscles precede those in the gastrocnemius muscles, which likewise precede changes in the centre of pressure.

DISCUSSION

In the present study, both the paraspinal and gastrocnemius muscles became activated in stereotyped patterns in response to a bipolar-binaural galvanic stimulus. The activation in both muscles preceded, and was consistent with, the changes in the COP. Initial activation of the ipsilateral paraspinal muscle and the contralateral gastrocnemius was followed by secondary activation of the contralateral paraspinal muscle and the ipsilateral gastrocnemius. This pattern

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Fig. 2. Representative paraspinal iEMG from one subject ensemble averaged across stimuli grouped by stimulus direction. Figure 2a shows the iEMG response of the left paraspinal muscles to anode left stimulation. Figure 2b shows the corresponding response in the right paraspinal muscle. Figures 2c and d show the left and right paraspinal muscle responses to anode right stimulation. In each of the figures, the stimulation period is shown in grey. Initial excitation, followed by inhibition can be seen in the ipsilateral muscles, whereas the opposite pattern is evident in the contralateral muscles.
of activation moved the body towards the anode and then maintained this deviated position. The opposite pattern of muscle activation and consequent postural sway was observed following the termination of the stimulus. The response of the paraspinal muscles to GVS has not been previously reported. Its relation to the gastrocnemius muscle response and the implications of this finding for the control of posture in the frontal plane will be discussed.

For the purpose of this discussion, an idealized model of the pattern of muscle activation was constructed in the following manner. The response was divided into two epochs (Table II and Fig. 7). Phases I and II are components of the initiation response. Phases III and IV are components of the termination response. Phase I begins with the initiation of the primary response (ipsilateral activation in the paraspinals and contralateral activation in the gastrocnemius). The end of phase I and the beginning of phase II occurs where the primary response peaks and the secondary response (contralateral activation in the paraspinals and ipsilateral activation in the gastrocnemius) begins. The response following the termination of the stimulus (Phases III and IV) is the opposite of the onset response. These response phases will be used throughout the remainder of the discussion.

**Pattern and timing of gastrocnemius muscle response**

Our findings regarding the pattern of activation in the gastrocnemius muscles and the corresponding centre of pressure are consistent with those of numerous authors (5, 6). Tokita (6) described two components of the soleus iEMG response to head forward binaural-bipolar GVS: i) an “initial response” in which the soleus muscles were activated on the side of the cathode and inhibited on the side of the anode occurred with a latency of 150 ms on the cathode side and 100 ms on the side of the anode. This initial response is analogous to phase I of our response; and ii) A “deviation response”, which is analogous to phase II in our observations. They also observed the transient deviation of the COP to the ipsilateral side, which was also found in our study, with a latency of 200 ms.

Day (5) also found latencies similar to those found in the present study. An initial response beginning at

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**Fig. 3.** Representative gastrocnemius iEMG from the same subject as in Figure 2. The placement of the graphs is the same as in Figure 2. A discernible pattern of muscle activity is not clearly seen in these figures.
approximately 120 ms was similar to our finding of $T_1 = 188$ ms. However, they found a second crossing of the superimposed tracings at approximately 420 ms, that was significantly shorter than our value of $T_3$ (511 ms). They considered the initial response to be the period between $T_1$ and $T_3$, whereas we have termed the period from $T_1$ to $T_2$ to be the initial response (Phase I). The choice of $T_1$ to $T_2$ for the first phase of the response makes more sense physiologically as this is the time during which only the contralateral gastrocnemius is activated. They did not describe the pattern of muscle activation that they observed in their study, thus it is not possible to determine whether the pattern of gastrocnemius activation that they obtained is in agreement with our findings. Their report of the centre of pressure is in agreement with our findings. However, as with the EMG, they only show the response to the onset of the stimulus, not the effect of its termination.

Table I. *EMG Latencies (mean ± SEM in ms):* $T_1$, $T_2$ and $T_3$ are measured from the time of the initiation of the stimulus. $T_4$ and $T_5$ are measured from the time of the termination of the stimulus

<table>
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<tr>
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<th>$T_1$ (ms)</th>
<th>$T_2$ (ms)</th>
<th>$T_3$ (ms)</th>
<th>$T_4$ (ms)</th>
<th>$T_5$ (ms)</th>
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<tr>
<td>Gastrocnemius</td>
<td>118 ± 17</td>
<td>262 ± 54</td>
<td>511 ± 37</td>
<td>156 ± 100</td>
<td>1358 ± 152</td>
</tr>
<tr>
<td>Paraspinal</td>
<td>74 ± 20</td>
<td>232 ± 27</td>
<td>643 ± 65</td>
<td>263 ± 140</td>
<td>1223 ± 202</td>
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Pattern and timing of paraspinal muscle response

The observed pattern of activation of the paraspinal muscles complemented that found in the gastrocnemius muscles but occurred slightly earlier. The timing of the activation in the paraspinal muscles is suggestive of descending control of postural musculature by the vestibulospinal system in response to a vestibular stimulus. The initial response in the paraspinal muscles was an activation of the ipsilateral side and an inhibition of the contralateral side, which occurred 74 ± 20 ms after the onset of the stimulus (vs 118 ± 18 ms for the onset of activity in the gastrocnemius). This pattern of activation (ipsilateral activation in the paraspinals and contralateral activation in the gastrocnemius) is consistent with the acceleration of the body towards the anode. The ipsilateral paraspinal muscle pulls the body towards the anode. The contralateral gastrocnemius assists in the movement by “pushing” the body in the same direction. This “push-pull” mechanism allows the body to use muscles along its ventral or dorsal surfaces to control motion in the frontal plane.

The second phase of the response in the paraspinal muscles began 232 ± 27 ms following the onset of the stimulus (vs 262 ± 54 ms in the gastrocnemius muscles). The coordinated action of the paraspinal and gastrocnemius muscles during Phase II serves to decelerate the body and maintain the offset position of
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the centre of mass. We were unable to quantify the timing of Phase III because of the observed variability in the dEMG at the end of phase II. The effect of the muscle activation during Phase III was the acceleration of the body back towards its neutral position (centred over the base of support) in response to the termination of the stimulus. Phase IV of the response in the paraspinal muscles began 263 ± 140 ms after the termination of the stimulus compared to 156 ± 100 ms in the gastrocnemius. It is not known why the secondary termination response begins in the gastrocnemius before it does in the paraspinals, although the difference between these two is not statistically significant. The result of Phase IV was the deceleration of the body as it neared the centre of its lateral base of support. The timing of this phase of the response is far more variable than the other phases. This is thought to occur because as the body returns to the centre of its base of support, no longer under the influence of the galvanic stimulus, it resumes its natural sway.

Table II. Description of phases of response employed in model of dEMG

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<tr>
<th>Stimulus</th>
<th>Phase</th>
<th>Description</th>
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<tr>
<td>During (onset response)</td>
<td>I</td>
<td>Primary response</td>
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<tr>
<td></td>
<td>II</td>
<td>Secondary response</td>
</tr>
<tr>
<td>After (termination</td>
<td>III</td>
<td>Primary response</td>
</tr>
<tr>
<td>response)</td>
<td>IV</td>
<td>Secondary response</td>
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Paraspinal and gastrocnemius muscles in the control of posture in the frontal plane

The results of this study show that: i) both the paraspinal and gastrocnemius muscles become activated in response to frontal plane galvanic stimulation, ii) the activation of the paraspinal muscles precedes the activation of the gastrocnemius, and iii) the pattern of activation is opposite in these two groups of muscles. This mode of response to frontal plane stimulation is reminiscent of the “hip strategy” that was reported by Horak and Nashner (10) in the response to platform perturbations in the sagittal plane. However, our data suggest that this movement occurs at the low back for the frontal plane. Day (5) showed similar responses occurred primarily between the torso and the pelvis, not the pelvis and the thigh for responses in the frontal plane. It thus makes sense to term this a “low back” strategy for the frontal plane response.

Muscles on the dorsal and ventral surfaces of the body are able to effect motion in the frontal plane by acting antagonistically (i.e. excitation of one muscle and inhibition of its contralateral partner). Conversely, synergistic action (simultaneous excitation or inhibition muscles on both sides of the body) of muscles on the dorsal and ventral surfaces of the body produces motion in the sagittal plane. In the present study, the initial response was the coordinated antagonistic action of the paraspinal muscles to accelerate the torso toward the anodal ear. The opposite pattern of antagonistic action in the gastrocnemius muscles enhanced the movement of the body. It is hypothesized that the gastrocnemius muscles are able to effect movement of the body in the frontal plane because the centre of mass is positioned ante-
rior to the ankle joint. However, a definitive answer to this question requires the simultaneous recording of the position of the body's centre of mass, which will be incorporated into future studies.

CONCLUSION

The paraspinal muscles have strong, distinct patterns of activation to transmastoid GVS with the head facing forward. This pattern is characterized by the initial activation of the ipsilateral muscle, followed by the activation of the contralateral muscle. The measurement of this response could prove useful in the clinical evaluation of vestibular nerve function. The postural sway response to GVS, as measured by the displacement of the COP, has previously been used clinically to detect the presence of intact vestibular nerve fibres (4) and to make the differential diagnosis between inner ear and retro-labyrinthine disorders of the vestibular system (3). The measurement of the paraspinal EMG yields the same information, but has the advantage of being more robust and more readily available in the clinical setting than measurement of the COP. Future studies of the responses of the paraspinal muscles to GVS need to be conducted in individuals with defined vestibular deficits to identify changes in the patterns of paraspinal muscle activation.

ACKNOWLEDGMENTS

This research was supported by The Scientific and Technical Research Council of Turkey.

REFERENCES


Submitted April 26, 1999; accepted September 16, 1999

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